geon and to the interventional cardiologist. Regarding the combination of MLR and conventional bypass operations, Allen and associates[1] recently presented favorable results of a multicenter prospective randomized study; therefore we agree that the combination of revascularization techniques using minimally invasive off-pump surgery and MLR (percutaneous or surgical) will have a role in the future for selected subsets of patients. This multimodality collaborative approach to myocardial revascularization is currently being applied increasingly at our institution, and we agree with Mack's recent forecast[2] that in 10 years 50% of all cardiac operations will be combined with a catheter-based procedure. To quote Rihal and Yusuf,[3] perhaps the relevant clinical question is not which mode of treatment is best, but which combinations of treatments, in what sequence, are appropriate for a specific patient at a specific point in his or her clinical course.

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12/8/101410

Aortic arch reconstruction without circulatory arrest

To the Editor:

We read with great interest the discussion of a modified perfusion technique during Norwood reconstruction by Pigula, Siewers, and Nemoto[1] and can offer some modifications that will further improve on this technique. In their report, the authors describe a technique to provide regional cerebral perfusion through the distal end of a partially constructed 3.5-mm polytetrafluoroethylene shunt to provide cerebral flow through the innominate artery during the "circulatory arrest" period. The authors monitor regional cerebral oxyhemoglobin saturation and relative cerebral blood volume index and demonstrate that the collateral network provides seemingly adequate cerebral blood flow while the arch is being reconstructed. Further supporting the presence of an extensive collateral network, the authors note the need for an aortic crossclamp on the distal aorta to prevent the collateral flow from flooding the operative field.

We have used a similar technique for Norwood-type reconstruction in hypoplastic left heart syndrome and describe some differences in technique that offer further benefit. In contrast to Pigula, Siewers, and Nemoto, our first maneuver after a short period of surface cooling (to 34°C), sternotomy, and heparinization is to create the proximal end of a 3.5-mm polytetrafluoroethylene shunt at the base of the right subclavian artery. This shunt is left long and is cannulated with the arterial inflow cannula (8F, Research Medical, Inc, Salt Lake City, Utah). Cardiopulmonary bypass is then initiated with bicaval venous cannulation and the pulmonary arteries are controlled with snares. The patient is cooled to 18°C over 20 minutes.

The advantage of this modification in technique is that a large portion of the procedure can then be performed during full cardiopulmonary bypass while cooling. Specifically, using the shunt as the arterial inflow leaves the aortic arch and ductus arteriosus free of cannulas. Consequently, the ductus can be ligated and divided, after which the main pulmonary artery can be divided and the pulmonary artery defect closed with a small polytetrafluoroethylene patch. Furthermore, the right atrium can then be opened and the atrial septectomy performed. There is no possibility of ejecting air in cases of aortic atresia due to the valve atresia and the fact that the main pulmonary artery is divided. By this time, the patient has been nearly completely cooled to 18°C. A "drop in" sucker can be left in the right atrium to keep the field clear.

Up to this point, the patient has been maintained on full cardiopulmonary bypass without any myocardial ischemia. Flow can be momentarily interrupted and cardioplegic solution infused through the arterial inflow cannula with the descending aorta clamped and the subclavian and carotid arteries temporarily occluded. Cardiopulmonary bypass is then resumed at 30 to 60 mL/min with the base of the innominate artery occluded and the distal subclavian and right carotid artery snares released. We monitor pressure in the right radial artery and maintain it at 20 to 30 mm Hg. The arch reconstruction is then completed and, like Pigula, Siewers, and Nemoto, we use a crossclamp on the distal aorta to avoid collateral flow from obscuring the operative field.

On completion of the arch reconstruction, the neo-aortic arch is cannulated with a second cannula and the arch deaired. Arterial inflow is then transferred to the new cannula and full cardiopulmonary bypass resumed. While the patient is being rewarmed, the distal end of the 3.5-mm shunt is trimmed and an anastomosis completed with the right pulmonary artery.

The chief advantage of these modifications is better time efficiency during the case. All the portions of the repair outside the aortic arch are performed during the cooling period with full cardiopulmonary bypass and without cardioplegic arrest. Myocardial ischemia and regional cerebral perfusion are limited to only the period during which the ascending aorta is open. This shortened period of systemic "circulatory arrest" aug-

mented with regional cerebral perfusion may provide better cerebral protection than more conventional techniques.

Although the data presented by Pigula, Siewers, and Nemoto are suggestive of adequate cerebral protection, we still believe that core cooling to 18°C is the principal component of our cerebral protection strategy. Further study is warranted to ascertain the relative merits of regional cerebral perfusion versus hypothermia for cerebral protection during aortic arch reconstruction in the neonate.

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12/8/101415

Drug therapy before coronary artery operations

To the Editor:

We were greatly interested by the results of the IMAGE trial,[1] which suggests European patients are more susceptible than US patients to aprotinin-induced vein graft occlusion. This has prompted us to inquire whether our patients have a European or US response to aprotinin.

We have used aprotinin in varying doses for patients undergoing redo operations or for those with recent aspirin ingestion. Table I describes our mortality rate in consecutive patients undergoing coronary artery bypass grafting with or without other procedures between 1993 and 1998; a dose-dependent increase in mortality is evident. However, the indications for aprotinin are also very important risk factors for perioperative mortality. Thus it may not be too surprising that patients given aprotinin have a higher mortality rate. To clarify this, we have further analyzed our data to take risk factors for mortality into account.

The patients and model have been described in detail elsewhere.[2] Logistic regression was performed with in-hospital mortality from any cause as the dependent factor and 22 independent factors: age, sex, urgency of the operation, history of cardiac surgery, concurrent cardiac or cardiovascular procedures, left ventricular function, recent myocardial infarct, left main coronary artery stenosis, chronic airways disease, cerebrovascular disease, diabetes, hypertension, and other risk factors including preoperative cardioactive drug use. Stepwise regression was not used.

In this model we found the relative risk of mortality attributable to aprotinin to be 1.34 per million units of aprotinin with 95% confidence intervals of 1.1 to 1.7 ($P = .01$ for relative risk = 1). This indicates a risk-adjusted association between aprotinin and in-hospital mortality.

In the jargon of evidence-based medicine, our data are "class III evidence" and should not be considered conclusive. However, taken in conjunction with the IMAGE trial results, our data support the hypothesis that in some circumstances aprotinin may have a deleterious effect on patients undergoing coronary artery operations.

Table I. *Dose of aprotinin, number of patients, and in-hospital mortality rate for 1593 consecutive patients undergoing coronary artery operations*

Dose of aprotinin (10^6 KIU)	*n*	*Mortality rate (%)*
0	839	1.8
<1	248	3.2
1-1.9	191	3.1
2-2.9	201	6.5
3-3.9	62	9.7
≥4	52	11.5

We do not understand why aprotinin appears to have adverse effects in some surgical units and not others. We use balanced salt solutions for vein distention, maintain kaolin-activated clotting times above 600 seconds, and never administer our blood cardioplegic solution through vein grafts. Until it becomes clear why the adverse effects of aprotinin appear to be site specific, we would suggest that aprotinin should be used with caution in coronary artery operations.

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12/8/101197

Reply to the Editor:

The letter by Weightman and Newman suggests a dose-dependant mortality for aprotinin doses ranging from 0 to ≥4,000,000 KIU (see their Table I). In the IMAGE study (J Thorac Cardiovasc Surg 1998;116:716-30), the average dose in aprotinin-treated patients was 5,550,000 KIU (n = 436). This total includes the 2,000,000 KIU loading dose, 2,000,000 KIU into the cardiopulmonary bypass circuit prime, plus 1,570,000 KIU by continuous infusion at a prescribed rate of 500,000 KIU per hour. IMAGE study results showed that this administration regimen for aprotinin had no effect on mortality (placebo, 1.6%; aprotinin, 1.4%). In the